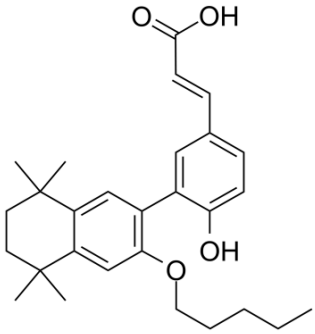
UVI 3003

Cat. No.:	HY-107500
CAS No.:	847239-17-2
Molecular Formula:	$C_{28}H_{36}O_4$
Molecular Weight:	436.58
Target:	RAR/RXR; Autophagy
Pathway:	Metabolic Enzyme/Protease; Autophagy
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (229.05 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2905 mL	11.4527 mL	22.9053 mL
5 mM	0.4581 mL	2.2905 mL	4.5811 mL
10 mM	0.2291 mL	1.1453 mL	2.2905 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.73 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.73 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

UVI 3003 is a highly selective antagonist of retinoid X receptor (RXR), and inhibits xenopus and human RXRα in Cos7 cells, with IC_{50}s of 0.22 and 0.24 μM, respectively.

IC_{50} & Target

IC50: 0.22 μM (Xenopus RXRα, in Cos7 cells), 0.24 μM (Human RXRα, in Cos7 cells)[1]

In Vitro

UVI3003 inhibits the activity of xenopus and human RXRα, with IC_{50}s of 0.22 and 0.24 μM, respectively. UVI3003 fully activates xPPARγ with an EC_{50} of 12.6 μM, and is almost completely inactive on hPPARγ and mPPARγ[1]. UVI 3003 (10 μM) does not change the proliferation rate of extraocular muscles (EOM)-derived or LEG-derived EECD34 cells. UVI 3003 causes a 65.4% difference in EECD34 cell fusion and desmin expression[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]

At ~30-40% confluence cells are treated with vehicle (ethanol), all-trans retinoic acid (1 μM), the RAR inverse agonist BMS493 (10 μM), or the RXR antagonist UVI 3003 (10 μM) for 24 h in proliferation media with a final concentration of ethanol at 0.1% for all treatments. At the end of the 24-h treatment cell proliferation rates are assessed[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Zhu J, et al. The unexpected teratogenicity of RXR antagonist UVI3003 via activation of PPARγ in Xenopus tropicalis. Toxicol Appl Pharmacol. 2017 Jan 1;314:91-97.

[2]. Hebert SL, et al. Effects of retinoic acid signaling on extraocular muscle myogenic precursor cells in vitro. Exp Cell Res. 2017 Dec 1;361(1):101-111.

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